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Association between carbohydrate intake and the risk of psoriasis: a prospective cohort study based on UK Biobank

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Abstract

Background Research on the association between carbohydrate intake and psoriasis risk is limited. We aimed to examine the associations of carbohydrate and its different subtypes with psoriasis risk, as well as the interaction between genetic predisposition and carbohydrate intake.

Methods We performed a prospective cohort study based on UK Biobank that included 210,474 participants who did not have psoriasis at baseline. A 24-hour dietary assessment tool was used to assess detailed dietary intake information. Incident psoriasis events were identified through hospitalization records. The association between carbohydrate intake and psoriasis was examined by Cox proportional hazard regression models. Multiplicative interaction between genetic risk and carbohydrate intake was assessed by incorporating a cross-product term in the model.

Results A total of 1907 incident psoriasis events were recorded during the follow-up period (median: 13.25 years). Compared to the lowest intake quartile (Q1), the highest intake quartile (Q4) of total sugars [HR (95% CI) = 1.14 (1.01–1.29), $FDR\text{-}P_{trend}$ = 0.116], free sugars [1.22 (1.07–1.38), 0.021], and sucrose [1.14 (1.01–1.30), 0.058] was associated with an increased psoriasis risk. In contrast, the highest intake of starch [0.86 (0.76–0.98), 0.049] and fiber [0.84 (0.74–0.96), 0.021] showed an inverse association with psoriasis risk. However, there was no statistically significant interaction between carbohydrate intake and genetic risk.

Conclusion Intake of total sugars, free sugar, and sucrose was positively associated with psoriasis risk, while fiber and starch were inversely associated.

Keywords Carbohydrate intake, Psoriasis, Cohort study, Association, Genetic risk

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Introduction

Psoriasis is a prevalent inflammatory skin disorder characterized by erythematous scaly plaques [1]. The incidence of psoriasis globally in 2019 was 57.8 per 100,000 people, and the prevalence of psoriasis was 503.6 per 100,000 people [2]. As a chronic disease, psoriasis significantly impacts the quality of life of affected individuals, both physically and psychologically [3]. Currently, the economic costs of psoriasis treatments are relatively high, leading to a significant economic burden [4]. Investigating the potential risk factors associated with psoriasis is essential for the prevention of the disease onset, thereby alleviating the burden on public health.

Previous epidemiological evidence suggested that omega-3 polyunsaturated fatty acids [5], vitamin D [6], and selenium [7] were associated with psoriasis risk, indicating that dietary nutrients play a role in the etiology of psoriasis. Carbohydrates are essential macronutrients with diverse dietary sources and a complex classification system. While carbohydrate intake has been linked to several autoimmune diseases, such as inflammatory bowel disease [8], multiple sclerosis [9], and rheumatoid arthritis [10], there are still limited studies relating to psoriasis. To date, only two epidemiological studies have explored the relationship between carbohydrate intake and psoriasis. Specifically, one cohort study suggested that sugar intake was inversely associated with psoriasis [11], and another case–control study indicated that dietary fiber may serve as a protective factor for psoriasis [12]. Therefore, a comprehensive investigation of the association between carbohydrate intake and psoriasis risk is needed.

To address the knowledge gaps mentioned above, our study aimed to investigate the association between carbohydrate intake and psoriasis risk, as well as the potential interaction between genetic susceptibility and dietary carbohydrate.

Methods

Study design and population

The UK Biobank (UKB) is an ongoing population-based prospective cohort study that has recruited over 500,000 individuals aged 39 to 72 from 22 sites across England, Scotland, and Wales between 2006 and 2010. At the baseline assessment, each participant contributed a diverse range of health-related data, encompassing demographics, lifestyle factors, and medical history, collected through touchscreen questionnaires and verbal interviews. The participants also provided body size measures and biological samples, and agreed to be followed up through linkage to their health records. Ethical approval for the UKB was obtained from the North West Multicenter Research Ethics Committee. All participants provided informed consent at baseline.

This study employed a prospective cohort design. Supplementary Fig. 1 presents the inclusion and exclusion flow. Initially, 210,946 participants who completed at least one 24-h dietary assessment were included. Then, after excluding 61 participants withdrawn from the cohort and 412 patients with prevalent psoriasis at baseline, a total of 210,474 participants were included in the main analysis. Furthermore, 4479 individuals whose genetic data were not available, 153 sex-mismatched individuals, 137 individuals with sex-chromosome aneuploidy, 31,974 individuals of non-Caucasian descent, and 52,135 genetically related individuals were excluded, resulting in a final inclusion of 121,596 individuals for analyzing interaction between genetic risk and dietary carbohydrate intake.

Dietary carbohydrate intake assessment

The Oxford WebQ is a web-based, 24-hour dietary assessment tool used to assess detailed dietary intake information (including 206 food items and 32 beverage types) [13]. In the UKB, dietary surveys were conducted in five rounds. The first round was carried out in person at assessment centers (April 2009 to September 2010). Participants with valid email addresses were then invited to complete four subsequent online survey rounds (Online Cycle 1: February 2011 to April 2011; Online Cycle 2: June 2011 to September 2011; Online Cycle 3: October 2011 to December 2011; Online Cycle 4: April 2012 to June 2012). Previous studies reported stable levels of food intake over four years following the baseline assessment in this cohort [14, 15]. Therefore, in our current study, we averaged participants' dietary intake data across multiple rounds to better reflect their habitual dietary intake. Nutrient intake was calculated by multiplying the quantity of portions consumed by the established portion sizes of each food or drink item, along with their respective nutrient composition. The nutrient information was obtained from the UK Nutrient Databank food composition tables [13].

The primary exposure variable was carbohydrate intake (including total sugars, starch, and fiber). Total sugars are classified based on their sources into free sugars and non-free sugars. Free sugars refer to all forms of added sugars (i.e., sugars added to foods by manufacturers, chefs, or consumers) plus the natural sugars found in honey, syrup, and unsweetened fruit and vegetable juices [16]. Non-free sugar intake was calculated by subtracting free sugar intake from total sugar intake [17, 18]. Non-free sugars primarily include the natural sugars found in fruits, vegetables, and dairy products [16]. Sugars can also be categorized by their chemical structure, which include monosaccharides (fructose and glucose) and disaccharides (lactose, maltose, and sucrose).

The residual method was used to calculate the energy-adjusted carbohydrate intake [19]. Additionally, we assessed the quartiles of energy-adjusted carbohydrate intake and categorized participants into four groups: the first quartile (Q1) (the lowest intake), the second quartile (Q2), the third quartile (Q3), and the fourth quartile (Q4) (the highest intake).

Ascertainment of psoriasis

The primary outcome was incident psoriasis, identified through UKB hospitalization records using International Classification of Diseases, Tenth Revision (ICD-10) codes L40.0-L40.9 [20]. We calculated follow-up time from the baseline assessment date until the earliest of: (1) psoriasis diagnosis date, (2) death date, (3) loss-to follow up date, or (4) study end date (31 October 2022).

Genetic risk score

The UK BiLEVE array and the UKB Axiom array were employed in the genotyping process. Comprehensive details about the genotyping process, imputation process, and quality control were provided in the previous study [21]. To calculate the genetic risk score (GRS) for psoriasis, we selected 61 single nucleotide polymorphisms (SNPs) from a published genome-wide association study (GWAS) conducted in a population of European ancestry [22] (Supplementary Table 1). Subsequently, the GRS was classified into three tertiles: the first tertile (T1, low genetic risk), the second tertile (T2, intermediate genetic risk), and the third tertile (T3, high genetic risk).

Covariates

We identified covariates based on the existing literature [23–25] and constructed a directed acyclic graph (Supplementary Fig. 2) using the online tool Dagitty (www.dagitty.net) to visualize the relationships between carbohydrate intake, psoriasis, and the selected covariates. The selected covariates included age (continuous), sex (female or male), race (white or non-white), the Townsend Deprivation Index (TDI) (continuous), body mass index (BMI) (continuous), education level (vocational qualification, any school degree, higher degree, or none of the above), alcohol drinking status (never, previous, or current), and smoking status (never, previous, or current). TDI is a composite measure derived from four socioeconomic indicators: unemployment rates, household overcrowding, non-car ownership, and non-home ownership. This validated metric is widely used in UK population studies, in which higher TDI scores indicate greater socioeconomic deprivation [26]. In the genetic analysis, we additionally adjusted for the top 10 principal components of ancestry (continuous) and genotyping batch (continuous).

Statistical analysis

All statistical analyses were performed utilizing R software version 4.4.1. Supplementary Table 2 details missing covariates, which we addressed using multiple imputation via the R mice package.

We presented normally distributed continuous variables as mean (standard deviation, SD), skewed variables as median (interquartile range, IQR), and categorical variables as count (percentage, %). Comparing incident psoriasis cases with non-psoriasis participants, we used Student's t-tests or Wilcoxon rank-sum tests for continuous variables and Chi-square tests for categorical variables. Cox proportional hazards models (follow-up time as time scale) were used to assess the association between energy-adjusted carbohydrate intake and psoriasis risk. The proportional hazards (PH) assumption was evaluated using Schoenfeld residuals. Model 1 included adjustments for sex and age, and Model 2 further adjusted for race, education level, TDI, BMI, alcohol drinking status, and smoking status based on Model 1. We treated quartiles as continuous to test linear trends, considering an FDR-corrected $P_{trend} < 0.05$ as significant [27]. To examine the potential dose–response relationship between energy-adjusted dietary carbohydrate and psoriasis risk, restricted cubic spline (RCS) curves were employed. The selection of knots was guided by Akaike's Information Criterion. Stratified analyses were also performed based on sex (female or male), age (≤ 60 years or > 60 years), and BMI (≤ 25 kg/m^2 or > 25 kg/m^2) subgroups. Additionally, we assessed the joint effect of energy-adjusted carbohydrate intake and GRS on psoriasis. Multiplicative interaction was assessed by incorporating the cross-product term in the Cox proportional hazards model.

To evaluate the robustness of our main findings, we performed several sensitivity analyses included: (1) excluding participants with missing covariates; (2) excluding participants diagnosed with psoriasis within the first two years of follow-up; (3) excluding participants with extreme energy intake (males: < 3347 kJ/d or > 17,573 kJ/d; females: < 2092 kJ/d or > 14,644 kJ/d) [17]; (4) limiting to participants with at least two dietary assessments; (5) additionally adjusting for type 2 diabetes (ICD-10 code: E11), hypertension (ICD-10 codes: I10-I13, I15), and hyperlipidemia (ICD-10 code: E78); (6) additionally adjusting for energy-adjusted intake of fat and protein; (7) using age as the time scale for variables violating the proportional hazards assumption; and (8) restricting to participants who reported their 24-hour recall as "fairly typical".

Results

The baseline characteristics of participants are presented in Table 1. We observed that demographic variables (including age, sex, race, TDI, BMI, and education

Table 1 Baseline characteristics

Characteristics	Energy-adjusted total carbohydrates (g/d)			
	Q1 (≤ 229.78)	Q2 (> 229.78 & ≤ 255.97)	Q3 (> 255.97 & ≤ 280.61)	Q4 > 280.61)
N	52,619	52,618	52,618	52,619
Age (years), mean (SD)	55.90 (7.81)	56.22 (7.85)	56.24 (7.93)	55.95 (8.20)
Sex, n (%)				
Female	26,771 (50.88)	30,762 (58.46)	30,860 (58.65)	27,585 (52.42)
Male	25,848 (49.12)	21,856 (41.54)	21,758 (41.35)	25,034 (47.58)
Race, n (%)				
White	50,785 (96.51)	50,568 (96.10)	50,399 (95.78)	49,042 (93.20)
Others	1642 (3.12)	1846 (3.51)	2047 (3.89)	3375 (6.41)
TDI, median (IQR)	−2.20 (−3.68, 0.25)	−2.37 (−3.75, −0.01)	−2.40 (−3.78, −0.11)	−2.27 (−3.70, 0.17)
BMI, kg/m^2, median (IQR)	26.70 (24.13, 29.77)	26.21 (23.73, 29.29)	26.04 (23.56, 29.09)	26.10 (23.62, 29.15)
Education level, n (%)				
Vocational qualification	2749 (5.22)	2669 (5.07)	2896 (5.50)	3170 (6.02)
Any school degree	19,645 (37.33)	20,177 (38.35)	20,046 (38.10)	20,021 (38.05)
Higher degree	26,142 (49.68)	25,206 (47.90)	24,901 (47.32)	23,791 (45.21)
None of the preceding groups	3852 (7.32)	4311 (8.19)	4543 (8.63)	5312 (10.10)
Alcohol drinking status, n (%)				
Never	554 (1.05)	1173 (2.23)	1922 (3.65)	3203 (6.09)
Previous	745 (1.42)	1250 (2.38)	1739 (3.30)	2688 (5.11)
Current	51,280 (97.46)	50,142 (95.29)	48,909 (92.95)	46,663 (88.68)
Smoking status, n (%)				
Never	24,854 (47.23)	29,244 (55.58)	31,818 (60.47)	32,864 (62.46)
Previous	21,850 (41.52)	19,145 (36.38)	17,348 (32.97)	16,300 (30.98)
Current	5789 (11.00)	4071 (7.74)	3330 (6.33)	3297 (6.27)
Energy, kJ/d, median (IQR)	8746.38 (7243.43, 10,505.83)	8057.23 (6696.65, 9524.93)	8054.99 (6757.13, 9494.54)	8737.04 (7327.41, 10,372.26)
Energy-adjusted total sugars (g/d), median (IQR)	96.20 (78.60, 113.22)	116.56 (100.91, 132.47)	131.05 (114.11, 148.03)	154.30 (131.80, 178.33)
Energy-adjusted free sugar (g/d), median (IQR)	46.91 (33.17, 61.85)	55.43 (42.36, 69.99)	60.96 (46.70, 77.12)	69.81 (50.77, 93.14)
Energy-adjusted non-free sugar (g/d), median (IQR)	45.24 (32.09, 60.28)	57.57 (43.93, 72.76)	66.24 (50.91, 82.72)	78.45 (58.80, 100.91)
Energy-adjusted starch (g/d), median (IQR)	106.72 (87.83, 124.01)	126.65 (110.93, 142.25)	136.27 (119.66, 153.09)	146.48 (125.54, 168.95)
Energy-adjusted fiber (g/d), median (IQR)	14.60 (11.81, 17.60)	16.88 (14.25, 19.78)	18.33 (15.47, 21.42)	20.14 (16.61, 24.06)
Energy-adjusted fructose (g/d), median (IQR)	19.96 (13.66, 26.75)	24.81 (18.45, 31.69)	28.81 (21.76, 36.28)	35.80 (26.50, 45.86)
Energy-adjusted glucose (g/d), median (IQR)	19.37 (14.28, 24.92)	23.55 (18.32, 29.31)	26.93 (21.00, 33.16)	32.85 (25.11, 41.62)
Energy-adjusted lactose (g/d), median (IQR)	10.75 (6.83, 15.04)	13.25 (9.22, 17.51)	14.40 (10.08, 18.94)	15.13 (10.27, 20.34)
Energy-adjusted maltose (g/d), median (IQR)	4.93 (2.91, 8.07)	5.52 (3.85, 7.67)	5.64 (4.07, 7.56)	5.53 (3.82, 7.51)
Energy-adjusted sucrose (g/d), median (IQR)	33.99 (24.64, 43.67)	43.19 (34.74, 52.54)	48.75 (39.63, 59.15)	56.62 (44.83, 71.59)

Abbreviations:BMI body mass index, *IQR* interquartile range, *SD* standard deviation, *TDI* Townsend deprivation index

level) were relatively evenly distributed across the Q1-Q4 of total carbohydrate intake. However, lifestyle factors showed contrasting patterns: both the proportion of current alcohol drinking and current smoking showed a gradual decrease with increasing quartiles of total carbohydrate intake.

During a median follow-up period of 13.64 years, 1907 incident cases of psoriasis were identified. The relationship between energy-adjusted dietary carbohydrate intake and psoriasis risk was illustrated in Table 2. In the age- and sex-adjusted model, participants with the highest intake of free sugar [HR (95% CI) = 1.19 (1.05–1.35), $FDR\text{-}P_{trend}$ = 0.014] and maltose [1.17 (1.03–1.33), 0.024] had a positive association with psoriasis risk compared to those with the lowest intake. Conversely, participants with the highest intake of non-free sugar [0.78 (0.68–0.89), < 0.001], starch [0.83 (0.73–0.94), 0.005], fiber [0.72 (0.64–0.82), < 0.001], fructose [0.77 (0.68–0.87), < 0.001], and glucose [0.79 (0.70–0.90), 0.006] demonstrated an inverse association with psoriasis risk compared to those with the lowest intake. However, in the fully adjusted model, the associations of non-free sugar, fructose, glucose, and maltose with psoriasis risk were no longer statistically significant. The associations of free sugar [HR (95% CI) = 1.22 (1.07–1.38), $FDR\text{-}P_{trend}$ = 0.021], starch [0.86 (0.76–0.98), 0.049], and fiber [0.84 (0.74–0.96),

Table 2 Hazard ratios (95% confidence intervals) for the associations between dietary carbohydrate intake and the risk of psoriasis

	Quartiles of energy-adjusted carbohydrates (g/d)				P_{trend}	$FDR\text{-}P_{trend}$	Per IQR increase
	Q1	Q2	Q3	Q4			
Total carbohydrates (g/d)							
Range	≤ 229.78	> 229.78 and ≤ 255.97	> 255.97 and ≤ 280.61	> 280.61			
Cases/total person-years	524/690710	469/691853	442/692741	472/689004			
Model 1[a]	1.00 (Ref.)	0.90 (0.80–1.02)	0.85 (0.75–0.97)	0.91 (0.80–1.03)	0.072	0.096	0.92 (0.88–0.98)*
Model 2[b]	1.00 (Ref.)	0.97 (0.86–1.10)	0.94 (0.83–1.07)	1.01 (0.89–1.15)	0.987	0.987	0.97 (0.92–1.03)
Total sugars (g/d)							
Range	≤ 100.83	> 100.83 and ≤ 122.78	> 122.78 and ≤ 146.60	> 146.60			
Cases/total person-years	500/691146	451/692725	436/692080	520/688358			
Model 1[a]	1.00 (Ref.)	0.90 (0.79–1.02)	0.87 (0.76–0.98)	1.03 (0.91–1.16)	0.789	0.789	1.00 (0.95–1.05)
Model 2[b]	1.00 (Ref.)	0.97 (0.86–1.10)	0.97 (0.85–1.10)	1.14 (1.01-1.29)*	0.053	0.116	1.04 (0.99–1.10)
Free sugar (g/d)							
Range	≤ 42.19	> 42.19 and ≤ 57.48	> 57.48 and ≤ 75.41	> 75.41			
Cases/total person-years	451/692006	459/693207	461/692447	536/686649			
Model 1[a]	1.00 (Ref.)	1.02 (0.90–1.17)	1.03 (0.90–1.17)	1.19 (1.05–1.35)*	0.008	0.014	1.08 (1.03–1.13)*
Model 2[b]	1.00 (Ref.)	1.08 (0.95–1.23)	1.09 (0.96–1.25)	1.22 (1.07–1.38)*	0.003	0.021	1.07 (1.02–1.12)*
Non-free sugar (g/d)							
Range	≤ 43.90	> 43.90 and ≤ 60.84	> 60.84 and ≤ 80.17	> 80.17			
Cases/total person-years	534/688450	485/691799	455/692527	433/691533			
Model 1[a]	1.00 (Ref.)	0.89 (0.78–1.00)	0.82 (0.72–0.93)	0.78 (0.68–0.89)*	< 0.001	< 0.001	0.90 (0.85–0.96)*
Model 2[b]	1.00 (Ref.)	0.98 (0.86–1.11)	0.94 (0.83–1.07)	0.91 (0.79–1.03)	0.111	0.175	0.97 (0.91–1.02)
Starch (g/d)							
Range	≤ 108.66	> 108.66 and ≤ 128.63	> 128.63 and ≤ 148.79	> 148.79			
Cases/total person-years	541/688319	477/691999	449/693320	440/690672			
Model 1[a]	1.00 (Ref.)	0.88 (0.78–1.00)	0.84 (0.74–0.95)	0.83 (0.73–0.94)*	0.002	0.005	0.91 (0.86–0.96)*
Model 2[b]	1.00 (Ref.)	0.93 (0.82–1.05)	0.88 (0.78–1.00)	0.86 (0.76–0.98)*	0.013	0.049	0.92 (0.88–0.97)*
Fiber (g/d)							
Range	≤ 14.20	> 14.20 and ≤ 17.39	> 17.39 and ≤ 20.95	> 20.95			
Cases/total person-years	577/687451	474/691424	424/693280	432/692154			
Model 1[a]	1.00 (Ref.)	0.81 (0.71–0.91)	0.71 (0.63–0.81)	0.72 (0.64–0.82)*	< 0.001	< 0.001	0.85 (0.80–0.90)*
Model 2[b]	1.00 (Ref.)	0.89 (0.79–1.01)	0.82 (0.72–0.94)	0.84 (0.74–0.96)*	0.004	0.021	0.92 (0.87–0.97)*
Fructose (g/d)							
Range	≤ 18.91	> 18.91 and ≤ 26.69	> 26.69 and ≤ 35.65	> 35.65			
Cases/total person-years	568/688086	466/691780	424/693522	449/690920			
Model 1[a]	1.00 (Ref.)	0.81 (0.72–0.92)	0.73 (0.64–0.83)	0.77 (0.68–0.87)*	< 0.001	< 0.001	0.89 (0.84–0.94)*
Model 2[b]	1.00 (Ref.)	0.90 (0.79–1.02)	0.85 (0.74–0.96)	0.91 (0.80–1.04)	0.095	0.174	0.96 (0.91–1.02)
Glucose (g/d)							
Range	≤ 18.63	> 18.63 and ≤ 25.11	> 25.11 and ≤ 32.58	> 32.58			
Cases/total person-years	551/688404	430/692114	477/693156	449/690635			
Model 1[a]	1.00 (Ref.)	0.77 (0.68–0.88)	0.85 (0.75–0.96)	0.79 (0.70–0.90)*	0.003	0.006	0.92 (0.87–0.97)*
Model 2[b]	1.00 (Ref.)	0.85 (0.75–0.96)	0.97 (0.85–1.10)	0.93 (0.82–1.06)	0.613	0.742	0.98 (0.93–1.04)
Lactose (g/d)							
Range	≤ 8.90	> 8.90 and ≤ 13.34	> 13.34 and ≤ 18.07	> 18.07			
Cases/total person-years	512/689629	418/691997	495/692027	482/690657			
Model 1[a]	1.00 (Ref.)	0.80 (0.71–0.92)	0.95 (0.84–1.07)	0.92 (0.81–1.04)	0.608	0.668	0.98 (0.93–1.04)
Model 2[b]	1.00 (Ref.)	0.83 (0.73–0.94)	1.00 (0.88–1.13)	0.97 (0.86–1.10)	0.674	0.742	1.00 (0.95–1.06)
Maltose (g/d)							
Range	≤ 3.68	> 3.68 and ≤ 5.44	> 5.44 and ≤ 7.64	> 7.64			
Cases/total person-years	464/692549	435/693517	447/691767	561/686476			
Model 1[a]	1.00 (Ref.)	0.95 (0.83–1.08)	0.97 (0.86–1.11)	1.17 (1.03–1.33)*	0.015	0.024	1.06 (1.03–1.09)*
Model 2[b]	1.00 (Ref.)	0.97 (0.85–1.10)	0.97 (0.85–1.11)	1.09 (0.96–1.24)	0.191	0.262	1.03 (1.01–1.06)
Sucrose (g/d)							
Range	≤ 34.52	> 34.52 and ≤ 45.21	> 45.21 and ≤ 57.51	> 57.51			

Table 2 (continued)

	Quartiles of energy-adjusted carbohydrates (g/d)				P_{trend}	$FDR\text{-}P_{trend}$	Per IQR increase
	Q1	Q2	Q3	Q4			
Cases/total person-years	497/691695	429/693189	443/692095	538/687330			
Model 1[a]	1.00 (Ref.)	0.88 (0.77–1.00)	0.91 (0.80–1.04)	1.11 (0.98–1.25)	0.079	0.096	1.07 (1.02–1.12)*
Model 2[b]	1.00 (Ref.)	0.94 (0.82–1.07)	0.99 (0.87–1.13)	1.14 (1.01–1.30)*	0.021	0.058	1.07 (1.02–1.12)*

[a] Model 1: adjusted for age and sex

[b] Model 2: model1 adjustments plus race, education level, TDI, BMI, smoking status, and alcohol drinking status

* : $P < 0.05$

Abbreviations: BMI body mass index, *CI* confidence interval, *HR* hazard ratio, *Ref.* reference, *TDI* Townsend deprivation index

0.021] with psoriasis risk remained significant. Additionally, we observed that total sugars [1.14 (1.01–1.29), 0.116] and sucrose [1.14 (1.01–1.30), 0.058] showed positive associations with psoriasis risk, although they did not exhibit statistical significance in the age- and sex-adjusted model. For the carbohydrate variables that were significant in the fully adjusted model, the HRs per IQR increase were as follows: 1.04 (0.99–1.10) for total sugars, 1.07 (1.02–1.12) for free sugar, 0.92 (0.88–0.97) for starch, 0.92 (0.87–0.97) for fiber, and 1.07 (1.02–1.12) for sucrose. To further examine the dose–response associations of these significant carbohydrates with psoriasis risk, we plotted RCS curves, as shown in Fig. 1. Only fiber intake exhibited a nonlinear association with psoriasis risk ($P_{nonlinear} = 0.014$).

Results of stratified analyses by sex, age, and BMI are shown in Supplementary Tables 3–5. We found that relationships between dietary carbohydrates and psoriasis risk were consistent across different subgroups of age and sex ($P_{heterogeneity} > 0.05$). However, there was heterogeneity in the association between starch intake and psoriasis risk among individuals with BMI ≤ 25 kg/m^2[HR (95% CI) = 0.68 (0.53–0.88), $P_{trend} = 0.002$] compared to those with BMI > 25 kg/m^2[0.92 (0.79–1.06), 0.191] ($P_{heterogeneity} = 0.009$).

Several sensitivity analyses demonstrated robustness of the above findings (Supplementary Table 6), except for the analysis conducted after excluding participants diagnosed with psoriasis during the first two years of follow-up. Specifically, no significant association was observed between total sugars and psoriasis.

The association between GRS and the risk of psoriasis is presented in Supplementary Table 7. A higher genetic risk score was linked to an increased risk of psoriasis, with a fully adjusted HR (95% CI) of 2.34 (2.00–2.73) for high genetic risk compared to low genetic risk, and 1.32 (1.11–1.57) for intermediate genetic risk versus low genetic risk. Furthermore, we explored the joint effect of carbohydrate intake and GRS on psoriasis risk, as illustrated in Fig. 2. Compared to participants with low genetic risk and low intake of total sugars, participants with high genetic risk and high intake of total sugars [HR (95% CI) = 2.91 (2.33–3.65)], free sugar [2.58 (2.08–3.20)], and sucrose [2.85 (2.29–3.55)] exhibited the highest risk of psoriasis. Additionally, compared to participants with low genetic risk and high starch intake, those with high genetic risk and low starch intake [2.85 (2.25–3.62)], as well as fiber intake [2.51 (2.00–3.14)], also had the highest risk of psoriasis. We did not find any interactions between these carbohydrates and GRS (all $P_{interaction} > 0.05$).

Discussion

Through this large prospective cohort study, we found that the intake of total sugars, free sugar, and sucrose was positively associated with psoriasis risk. In contrast, the intake of starch and fiber was associated with a reduced risk of psoriasis. Participants at high genetic risk who also had high intake of total sugars, free sugar, and sucrose exhibited the highest risk of psoriasis, while those with low genetic risk and high intake of starch and fiber showed the lowest risk. However, no significant interactions between these carbohydrates and GRS were observed.

Our study found that total sugars may serve as a risk factor for psoriasis. Inflammation plays a significant role in the development of this condition [28]. A cross-sectional study demonstrated correlations between dietary sugar intake and inflammatory markers [29], suggesting that higher sugar consumption may increase systemic inflammation. Additionally, previous evidence has indicated that high sugar intake is a predominant characteristic of the Western diet [30]. An animal study showed that short-term adherence to a Western diet could exacerbate psoriasis-like skin inflammation [31]. Furthermore, we also found that sucrose, a type of disaccharide, was positively linked to psoriasis risk. An animal study also demonstrated that a high-sucrose diet increased inflammation, thereby accelerating the progression of rheumatoid arthritis [32]. On the other hand, sugars could be categorized as free sugar and non-free sugar based on food sources. A previous study has shown that patients with active systemic lupus erythematosus consumed significantly more free sugar than those with inactive disease [33]. Similarly, frequent consumption of sugar-sweetened

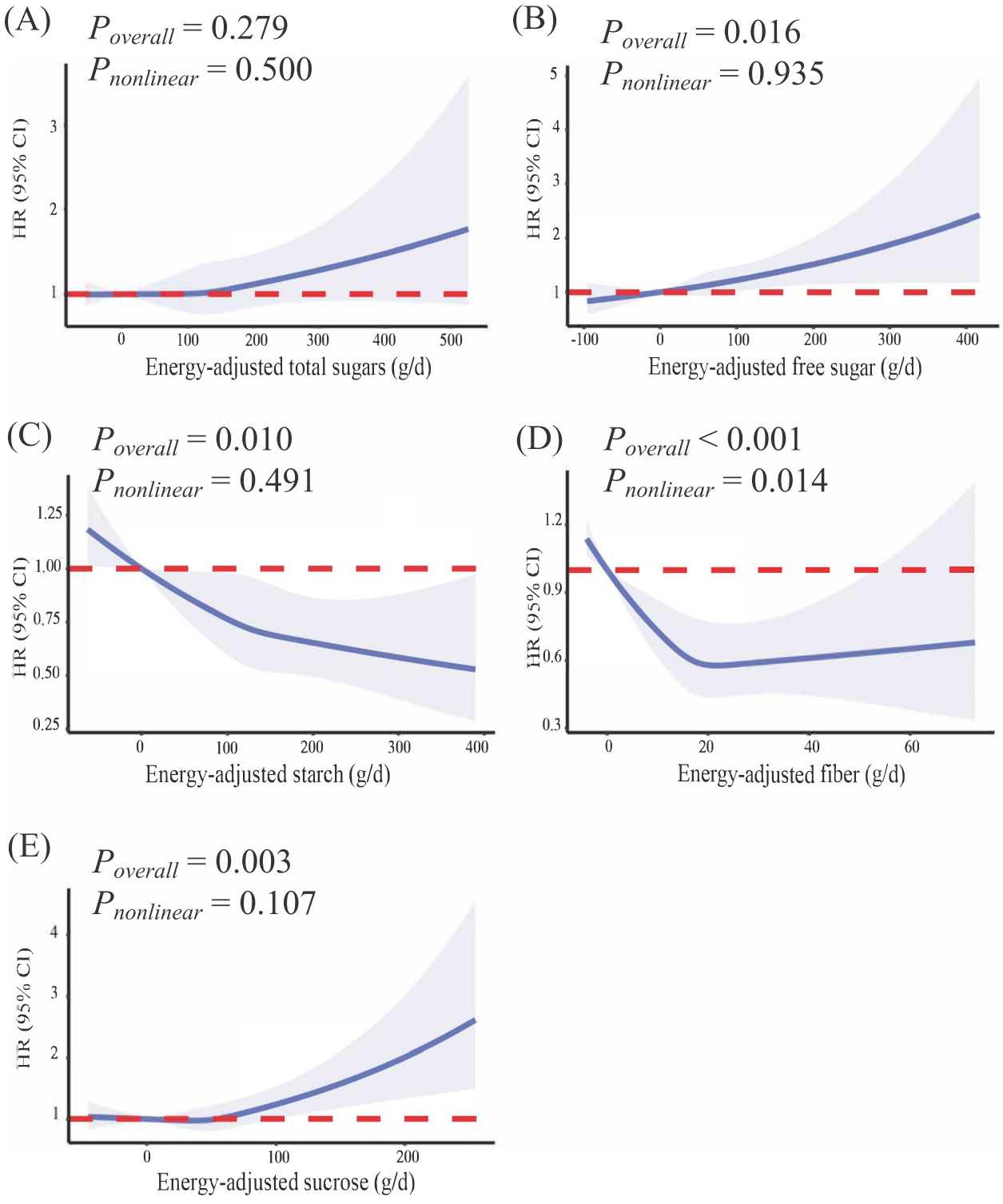

Fig. 1 Dose–response relationship of energy-adjusted total sugars (**A**), free sugar (**B**), starch (**C**), fiber (**D**), and sucrose (**E**) with psoriasis risk. The adjusted covariates included age, sex, race, education level, TDI, BMI, smoking status, and alcohol drinking status. The x-axis represents the energy-adjusted carbohydrate intake, while the y-axis displays the hazard ratio (95% confidence interval) for psoriasis risk. Abbreviations: BMI = body mass index; CI = confidence interval; HR = hazard ratio; IQR = interquartile range; TDI = Townsend deprivation index

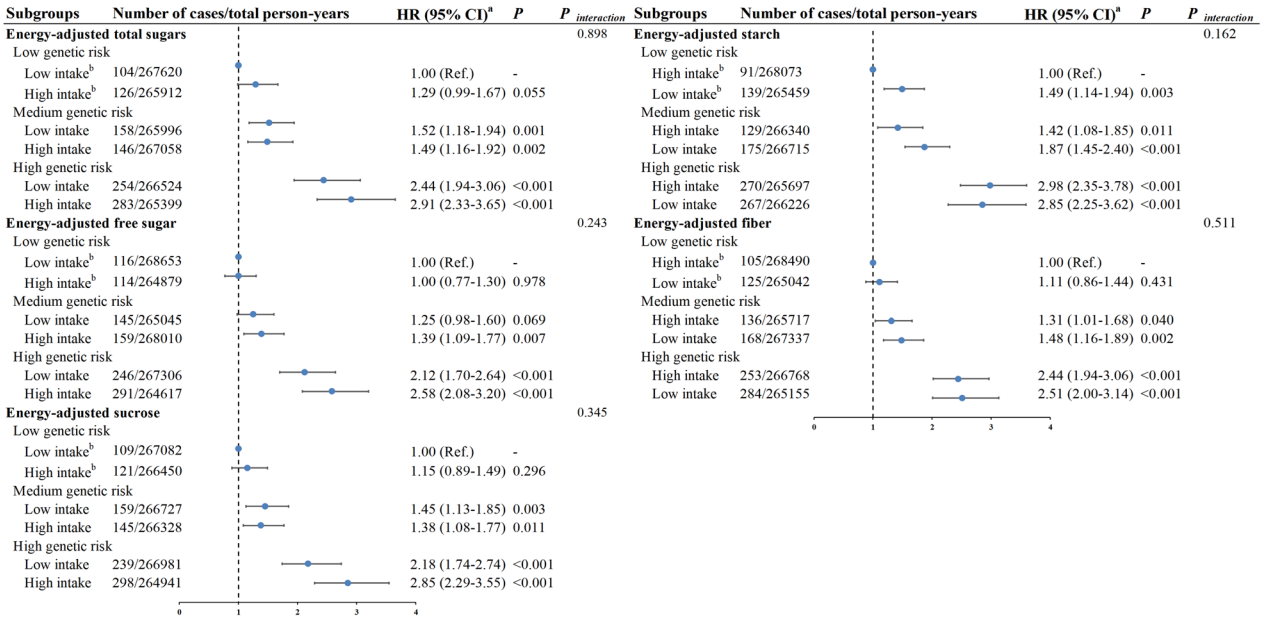

Fig. 2 Joint effects of energy-adjusted carbohydrate intake and genetic risk score on psoriasis risk. The adjusted covariates included age, sex, race, education level, TDI, BMI, smoking status, alcohol drinking status, the top 10 principal components of ancestry, and genotyping batch. Free sugar analyses used low genetic risk score/low intake as reference group, and starch and fiber analyses used high genetic risk score/low intake as reference group. [a] Adjusted for age, sex, race, education, TDI, BMI, smoking status, alcohol drinking status, the top 10 principal components of ancestry, and genotyping batch. [b] Energy-adjusted carbohydrates were divided into high and low intake levels based on the median (g/d) as follows: total sugars (≤ 122.73 vs. > 122.73), free sugar (≤ 57.56 vs. > 57.56), starch (≤ 128.50 vs. > 128.50), fiber (≤ 17.39 vs. > 17.39), and sucrose (≤ 45.00 vs. > 45.00). Abbreviations: BMI = body mass index; CI = confidence interval; HR = hazard ratio; IQR = interquartile range; Ref. = Reference; TDI = Townsend deprivation index

beverages, which are high in free sugar, has been linked to an increased risk of rheumatoid arthritis in women [10]. While prior studies have explored the relationships between sugar intake and autoimmune diseases, research on sugar's association with psoriasis—another classic autoimmune condition—remains limited. Our study indicated that free sugar was associated with an increased risk of psoriasis, helping to clarify this relationship and underscoring the need for further investigation.

In addition, our study demonstrated a negative association between dietary fiber intake and psoriasis risk. The Mediterranean diet is characterized by high consumption of fruits, vegetables, whole grains, and nuts, all of which are rich in dietary fiber [34]. A cross-sectional study revealed a negative correlation between the severity of psoriasis and adherence to the Mediterranean diet, suggesting that dietary fiber may mitigate the progression of psoriasis [35]. Mechanistically, dietary fiber is recognized for its anti-inflammatory properties. For example, a randomized controlled trial showed that a high-fiber diet contributed to the reduction of inflammation levels in obese and overweight women [36]. Given the pivotal role of inflammation in the pathogenesis of psoriasis [37], we hypothesize that increased dietary fiber intake or adoption of a fiber-rich dietary pattern may represent a promising nutritional intervention for psoriasis risk reduction, primarily through its anti-inflammatory effects.

Starch can be classified into three types: rapidly digestible starch (RDS), slowly digestible starch (SDS), and resistant starch (RS) [38]. RDS quickly releases glucose into the bloodstream, characterized by a high glycemic index (GI), with representative foods like potatoes and bread [39]. In contrast, SDS has a slower digestion time, leading to a gradual and sustained rise in blood sugar, characterized by a low GI, with examples including millet and legumes [38]. Previous studies indicated that high-GI diets increased the risk of inflammatory diseases [40, 41], while low-GI diets had the opposite effect [42, 43]. Additionally, RS is a starch component that cannot be broken down by digestive enzymes; it enters the colon directly, where gut microbes ferment it to produce short-chain fatty acids (SCFAs) with anti-inflammatory properties [44]. Thus, starch is a carbohydrate closely linked to inflammation, and its regulatory effects on inflammation also vary by type. In the current study, we found that higher starch intake was associated with a lower risk of psoriasis. This association may result from the combined effects of the three types of starch, with SDS and RS potentially playing a dominant role. However, since the UKB did not provide information on the types of starch consumed, we cannot fully explain this hypothesis, and further research is needed. Moreover, it is worth noting that some foods, such as whole grains and legumes, are rich in both starch and dietary fiber, which may also

contribute to the observed simultaneous protective effects of starch and fiber against psoriasis.

From a public health perspective, our findings suggest that population-level dietary modifications—particularly reducing intake of free sugar while increasing consumption of fiber-rich foods, especially among genetically high-risk individuals—could serve as an effective strategy for psoriasis prevention. This also provides a scientific foundation for future research aiming at developing precision nutrition approaches for psoriasis management in populations with different genetic risks.

Our study had the following strengths. First, there has been limited existing research on the relationship between carbohydrate intake and psoriasis. We conducted a large-sample prospective cohort study, providing novel evidence for this field. Second, we considered genetic factors and investigated the combined effects and interaction between genetic risk and carbohydrate intake. However, this study also had several limitations. First, the participants were from the UK, which limited the generalizability of the findings to other populations. Second, although PH assumption violations for total carbohydrates, starch, and lactose could introduce bias, age-time-scale sensitivity analyses showed an acceptable impact. Third, similar to other nutritional epidemiological studies, 24-hour dietary recall is inevitably subject to recall bias. Moreover, compared to food frequency questionnaires (FFQs), 24-hour dietary recall is less effective in assessing participants' long-term dietary habit. Therefore, the findings of this study require further validation through future studies using FFQ-collected data . Finally, while this study employed a cohort design, it remains observational in nature, and we cannot completely rule out reverse causation. Our sensitivity analyses—excluding participants diagnosed with psoriasis within the first two years of follow-up—revealed that the associations between sucrose/total sugars and psoriasis lost statistical significance. This attenuation may indeed reflect the diagnostic time lag, which complicates accurate classification of cases as prevalent versus incident psoriasis. These findings suggest the need for longer-term follow-up studies to determine whether the observed associations remain stable over time.

Conclusion

In summary, this study found that psoriasis risk was negatively associated with the intake of starch and fiber, while positively associated with the intake of total sugars, free sugar, and sucrose. Our research provided a theoretical basis for preventing psoriasis, thereby contributing to formulating informed public health policies.

Glossary

BMI	body mass index
CI	confidence interval
FDR	false discovery rate
FFQs	food frequency questionnaires
GI	glycemic index
GRS	genetic risk score
GWAS	genome-wide association studies
HR	hazard ratio
ICD-10	International Classification of Diseases, Tenth Revision
IQR	interquartile range
PH	proportional hazards
RCS	restricted cubic spline
RDS	rapidly digestible starch
Ref.	reference
RS	resistant starch
SCFAs	short-chain fatty acids
SD	standard deviation
SDS	slowly digestible starch
SNPs	single nucleotide polymorphisms
TDI	Townsend deprivation index
UKB	UK Biobank

Supplementary Information

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Supplementary Material 1.

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Institutional Review Board Statement
The UKB study was conducted in accordance with the guidelines of the Declaration of Helsinki and was approved by the North West Multi-Centre Research Ethics Committee (#21/NW/0157, 29 June 2021).

Authors' contributions
Conception and design: J.L. and Y.M.; formal analysis: H.L. and Y.Y.; visualization: H.L., L.W. and C.H.; writing original draft: H.L., J.L. and Y.M.; writing review and editing: H.L., J. L., Y.M, D.Y, X.S, and J.G. All authors mentioned above made substantial contributions to the content of the paper and approved the final version of the manuscript.

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Data availability
Data will be made available on request.

Declarations

Ethics approval and consent to participate

Written informed consent was obtained from the patient(s) to publish this paper.

Competing interest

The authors declare no competing interests.

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